

Lead abatement and prevention of developmental disabilities¹

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There are many causes of developmental disabilities including environmental factors. This article discusses "safe" levels of lead in children, and their association with developmental disabilities. Outcomes of this review of literature are that the current "safe" level of 10 ug/dl needs to be reconsidered, and that there is a need for continuing research in the area of lead pollution in Australia. Lead pollution in the north Lake Macquarie area of New South Wales (NSW) has long been of great concern to the public and for environmentalists. Data on blood lead level (Pb-B) of pre-school children in Australia and the north Lake Macquarie area, and the adverse health effects of lead for young children are also reviewed in this article. Although the trend of Pb-B for pre-school children in north Lake Macquarie has decreased, the percentage of children with Pb-B > 10 µg/dl over the last several years has been high and there is in fact no safe level for Pb-B. It is clear that a lead abatement strategy in the area is necessary. More studies are also required to investigate the relationship between low level lead exposure and the formation of developmental disabilities, especially in areas where lead pollution remains a concern. Lead toxicity is a major public health issue and present data limitation should not be an excuse for delay in the initiation of lead abatement strategies.

A general intervention for preventing developmental disabilities (DD) would be to understand the various causes of DD and subsequently to develop strategies and methods to eliminate them. Known causes of DD are many (Fils, 1980). However, half or even more of the causes remain unknown (Hattersley, Hosking, Morrow, & Myers, 1987). Nagai (1986) suggested that DD are results of a continuous interaction between genetic and environmental factors to the embryo, fetus, and infant. Guthrie and Young (1987) stated:

Among the thousands of causes known and unknown of mental retardation and developmental disabilities, environmental agents are possibly more important

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than genetic. Among these, our attention is increasingly drawn to the many products of modern industry that pollute our environment. Lead is perhaps the oldest of these. (p.163)

LEAD POLLUTION IN NEWCASTLE AREA, NEW SOUTH WALES

Newcastle is a major Australian industrial city and has, since the time of European settlement, been a major focus of the coal mining and metals industry. It has for many decades been perceived as a city polluted by smog and industrial pollutants. Although there has been an awareness of the need to decrease industrial pollution in Newcastle since 1947 (Newcastle City Council, 1972), air, water and soil pollution has remained a major concern for environmentalists, medical and public health professionals, politicians, and the public. In recent years, the problem of lead pollution and in particular, its specific adverse health effects on young children in Lake Macquarie which is adjacent to the Newcastle area, has been of great concern, especially in relation to lead pollution generated from the operation of the metals smelter in Boolaroo, a suburb of north Lake Macquarie.

Although there is evidence which demonstrates blood lead levels (Pb-B) of children in north Lake Macquarie have fallen, 43% of pre-school children (0 to 5 years of age) tested in 1994, still had blood lead level readings above 10 mg/dl² (NSW Parliament, 1994). Needleman and Gatsonis (1990), and Needleman, Schell, Bellinger, Leviton, and Allred (1990) were able to demonstrate that long-term low lead exposure in children had harmful health effects during their developmental stages. In fact, it has been found that Pb-B as low as 10 mg/dl can harm children (CDC, 1991). More importantly, as Piomelli (1994) has argued, it is accepted that there is no threshold for the effects of lead in the body, and research carried out by Needleman (1991a), has also confirmed that no safe level has yet been found for children.

ADVERSE HEALTH EFFECTS AND TOXIFICATION OF LEAD TO YOUNG CHILDREN

There is a vast body of literature concerning adverse health effects of lead on young children particularly in the United States (Garnys, Freeman, & Smythe, 1979; Needleman, 1979; Bellinger, Needleman, Leviton, Waternaux, Rabinowitz, & Nichols, 1984; Bellinger, Leviton, Needleman, Waternaux, & Rabinowitz, 1986; Dietrich, Krafft, Shukla, Bornschein, & Succop, 1987; Schroeder & Hawk, 1987; McCloy, 1989; Marino, Landrigan, Graef, Nussbaum, Bayan, Boch, & Boch, 1990; Needleman & Gatsonis, 1990; Needleman, Schell, Bellinger, Leviton, & Allred, 1990; Needleman, 1991a; Needleman, 1991b; Benson & Lane, 1993; Dyer, 1993; Gellert, Wagner, Maxwell, Moore, & Foster 1993; Yoder, Burright, & Donovanick,

²The "safe" level of Pb-B of pre-school children was decreased to 10 g/dl in June 1993 by the National Health and Medical Research Council (Berry, Garrard, Greene, Crooks, NIEIR, NETRU, Hallebone, Townsend, Braaf & Forster, 1994).

1993; Alperstein & Vimpani, 1994; Commonwealth Environment Protection Agency, 1994; Piomelli, 1994; and Needleman, Riess, Tobin, Biesecker, & Greenhouse, 1996). Lead is an extremely toxic metal which binds to a protein in the human body, and the developing brain and nervous system of young children are most susceptible to its toxic effects (Patterson, 1965; Sweeney, 1992; Benson & Lane, 1993; Piomelli, 1994). Lead has a long half-life, in excess of 20 years, and once absorbed, it is potent and persistent within the human body (Sweeney, 1992). A study performed by Garnys et al. (1979) in New South Wales showed that behavioural problems in schoolchildren were significantly associated with their lead burden, and this finding was consistent with overseas studies. A recent study in the United States found that children with high lead levels in their bones were more likely to engage in aggressive and delinquent behaviours than those with low levels, and these effects followed a developmental course (Needleman et al., 1996). Needleman et al. (1979), as well as Environmental Protection Authority NSW (EPANSW) (1994), identified a decrease in IQ points on average in children attributed to lead ingestion. Bergoni, Borella, Fantuzzi, Vivoli, Sturloni, Cavazutti, Tampieri, and Tartoni (1989), Lyngbye et al. (cited in DeRienzo-DeVivio, 1992), and Needleman (1991b) also found a strong link existing between low lead level exposure and lower IQ scores. A study of 1265 New Zealand children led Fergusson, Horwood, and Lynskey (1997, p. 471) to conclude that, "early mildly elevated lead levels have modest but detectable effects on individual achievement, with these effects extending to late adolescence". The mean denture lead level of the cohort in this study was only 6 µg/dl compared to the higher mean of 14 µg/dl in Needleman et al.'s (1990) sample.

In addition to the above findings, a common pathological effect in children of widespread environmental lead contamination is encephalopathy (Piomelli, 1994). It is commonly known that this medical condition can lead to DD. Lead readily crosses the placenta, placing the developing fetus at risk for neurological impairment, low birth weight, and low gestational age (McCloy, 1989; DeRienzo-DeVivio, 1992; Benson & Lane, 1993; and Commonwealth Environment Protection Agency, 1994). Hoffman (1990) argued that prolonged exposure to lead can affect both women's and men's reproductive systems, which increases the risk of birth defects in years to come. Developmental lead exposure also damages the visual cortex which leads to visual deficits (Boyes, 1992). A study performed by Perlstein and Attala (1966) found that 22% of a sample of 425 clinically lead-poisoned children developed intellectual disability, and 20% developed seizures. Studies performed by Bellinger, et al. (1984; 1986) indicated that babies who had higher cord Pb-B (up to 14.6 µg/dl) had significantly associated with lower Bayley Mental Developmental Index (MDI) scores. Among these babies, a cord Pb-B greater than 10 µg/dl was associated with early developmental disadvantage.

In brief, there is evidence which demonstrates that levels of lead exposure in childhood are associated with the occurrence of DD and behavioural problems. There is a need to further understand the toxic effects of Pb-B below 10 µg/dl in young children in Australia, and reconsideration of the perceived current safe Pb-B of ≤ 10 µg/dl and appropriate lead abatement program may be necessary.

EPIDEMIOLOGY OF LEAD POISONING OF PRE-SCHOOL CHILDREN IN AUSTRALIA, AND NORTH LAKE MACQUARIE

While data on lead exposures in the Australian population are finite except for the information for some lead towns, data on the extent of many lead sources in Australia are even more limited. It was formerly thought that adverse health effects which required attention and evaluation were associated with Pb-B of 25 µg/dl (Berry et al., 1994; Newcastle Environmental Toxicology Research Unit, 1994). Nevertheless, for this level, there is still no accurate epidemiological information about the numbers of affected pre-school Australian children, and only an estimate of between one and three percent of them was believed to be affected (Alperstein & Vimpani: 1994; Berry et al., 1994; Newcastle Environmental Toxicology Research Unit, 1994). In 1993, Environmental Protection Authority NSW and NSW Health Department estimated that there were 8,000 to 21,000 pre-school children (2-3.5% of the population) who had Pb-B above 25 µg/dl, and 57,000 to 162,000 (14-40%) who had levels above 15 µg/dl in NSW. The level that requires attention was reviewed and lowered by the National Health and Medical Research Council (NHMRC) in June 1993 to 10 µg/dl (Berry et al., 1994). Nonetheless, this lowered level is still found in an estimated 45% of Australian children (Berry et al., 1994).

In the suburbs of north Lake Macquarie which are located in close proximity to a lead smelter in Boolaroo, a multi-phased Pb-B monitoring of pre-school children by NETRU found that in 1994, 43% of the sample had Pb-B > 10 µg/dl, although the trend is decreasing and the mean is 10.4 µg/dl (NSW Parliament, 1994, Table 1). This result is alarming, and calls for prompt lead abatement actions, since it is believed that even a low Pb-B of below 10 µg/dl is still associated with detrimental developmental effects on children.

Table 1

Results of blood lead monitoring of children in north Lake Macquarie

	Phase 1 1991	Phase 2 1991	Phase 3 May 1993	Phase 4 Nov. 1993	Phase 5 May 1994
n	313	357	452	272	270
mean (µg/dl)	13.6	11.1	11	9.7	10.4
std deviation (µg/dl)	5.5	5.7	5.1	4.9	4.6
% ≥15 µg/dl	38	21	19	14	11
% ≥10 µg/dl	75	50	55	35	43

Source: NSW Parliament (1994). Report of the Select Committee upon Lead Pollution, Vol. 1 (p.29)

CURRENT PROGRESS IN CONTROLLING THE PROBLEM

In New South Wales, there are currently many committees, organizations, and departments which have been established to investigate, provide suggestions and/or implementation programs to address the lead pollution problem in north Lake Macquarie. These include the New South Wales Interdepartmental Lead Task Force and its nine

Interdepartmental Working Groups for different particular lead issues, the Environmental Lead Management Committee of PHU of HAHS, the Lake Macquarie Community Advisory Committee, the North Lakes Environment Action Defence Group Inc (No Lead), the EPANSW, NSWHD, and the Commonwealth Environment Protection Agency. All of these bodies have provided and published many and varied recommendations. However, as Berry et al.(1994) argued, there is a need to develop a well-coordinated strategy for reducing lead in the environment. Approaches to lead abatement in Australia have been largely ad hoc, but one major achievement is the reduction of lead in petrol which is regarded as the most significant source of lead exposure. However, it is estimated that even the radical removal of lead in petrol will still leave about 5% of children receiving significant exposure from other sources, for instance, lead containing household paint, which was widely used in the construction of pre-1950s dwellings and which is known to have a residual effect (EPANSW and NSWHD, 1993).

STRATEGIES FOR INTERVENTION

Although universal mass screening of Pb-B in Australian children cannot be justified on economic grounds, targeted screening in high risk areas is appropriate and will provide a basis for judging the effectiveness of current interventions (Berry et al., 1994). Screening is therefore suggested for all pre-school children in north Lake Macquarie to confirm and update the current situation of the problem and for possible further action. Studies in low Pb-B ($<10 \mu\text{g/dl}$) and DD of children in Australia are required, and the north Lake Macquarie area would be an appropriate area for this research. Pilot investigation of Pb-B of young children with DD who reside in large residential settings in Newcastle could also be carried out in order to obtain an initial insight about the relationship between Pb-B and DD.

Regarding lead contamination from paint, it is also suggested that buildings constructed before 1950 or at risk of lead contamination in Lake Macquarie be identified and inspected. It would be necessary for a register to be created and updated by local government. Subsequently householders and people responsible for children in these affected buildings should be provided with assistance to abate the lead contamination in their environment. It is suggested that local government establish a special department or section responsible for this type of assistance, with the cooperation of the Department of Housing. Additionally, children in the area who have double exposure to lead from the building and who live closer to the lead emission sources would be given first priority for Pb-B screening. For children already poisoned, treatment, rehabilitation, and avoidance of recurrence would be the immediate intervention. Again, this could be supported and assisted by the proposed special departmental section.

CONCLUSION

Lead pollution has been a long term problem in the Newcastle area and especially in north Lake Macquarie. While some progress has been made in attempts to eliminate

the problem of lead exposure, it remains of concern since research has shown that even low level exposure ($\text{Pb-B} \leq 10 \mu\text{g/dl}$) is considered to be harmful to young children. There is evidence to suggest an association between DD and low level of lead exposure as early as the beginning of pregnancy. In Australia, more studies are needed to investigate this issue and especially for local industrial areas where pollution remains a concern. Nevertheless, as Berry et al. (1994) argued, data limitation should not provide an excuse for delay. As Schroeder (1987) stated, professionals in the area of developmental disability must get involved now or abdicate their role in shaping public policy in lead abatement. This is of crucial concern in the primary prevention of developmental disability in Australian children.

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